165346

Reprinted from the Archives of Environmental Health
March 1970, Volume 20

Vaporized Formaldehyde Treatment of a Textile Mill Contaminated With *Bacillus anthracis*

Lowell S. Young, MD; James C. Feeley; and Philip S. Brachman, MD, Atlanta

Contamination with *Bacillus anthracis* spores is a serious environmental hazard in textile mills where imported raw goat hair is used. While a plant in which haircloth had been produced was being converted for synthetic fiber production, formaldehyde vapor was introduced into the sealed buildings at a final concentration of 1.38 to 1.62 ml/cu ft (18 to 21 mg/liter). Pretreatment rates of surface contamination with anthrax spores were 37% in the initial processing area and 12.5% in the spinning area. Contamination dropped to 8% and 1% immediately after formaldehyde treatment, and to 1% and 0 six months later. Test suspensions of *B anthracis* spores placed in the plant before it was treated showed 99.998% loss of viability and recovery of all bacterial flora had been reduced tenfold to 100-fold after treatment.

During the past 20 years in the United States, most cases of cutaneous or inhalation anthrax in humans occurred in association with textile mills in which raw goat hair imported from countries where anthrax is enzootic was utilized.[1] Goat hair processing causes extensive environmental contamination with anthrax spores, especially in the early stages of manufacturing operations. The initial processing areas are where anthrax spores are most frequently recovered from surfaces and where most of the persons in whom anthrax developed worked.[2]

The introduction of an experimental human anthrax vaccine for goat hair textile mill workers during the four years 1959 to 1962 reduced the annual incidence of human anthrax in the United States to only a few cases per year.[1] However, the environmental hazard obviously persists in these mills, necessitating annual booster immunizations for all who work in them and occasionally posing a lethal threat to persons who live or work nearby.[3]

One mill in the Southeast that had produced hair cloth exclusively from goat-hair fiber and where numerous cases of human cutaneous anthrax had occurred suspended operations in April 1967. Eight months later a new management decided to produce synthetic carpet yarns. Environmental sampling programs and experimental inhalation studies with primates carried out at this plant[4] before 1966 had shown that it was heavily contaminated with *B anthracis*. Extensive cleaning and renovating operations were planned because of the known persistence of spores of this organism. The Bacterial Diseases Section, Epidemiology Program, National Communicable Disease Center (NCDC), recommended gaseous disinfection with formaldehyde vapor, preceded and followed by environmental sampling studies for *B anthracis*, as a principal step in the renovating operations. Thus, the effectiveness of this technique with large industrial areas could be measured, and data on the persistence of anthrax organisms would be used to make recommendations on continuing anthrax immunization.

Nature of the Cleaning and Decontamination Operations and Survey Dates

The mill is similar to the plant described by Dahlgren et al.[3] There are three principal manufacturing areas, consisting of three separate brick buildings for (1) carding, where fibers are cleaned, sorted, and drawn into a thick loose rope, (2) spinning, where this rope is spun into threads and wound

Submitted for publication June 27, 1969; accepted Aug 19.

From the Epidemiology Program, National Communicable Disease Center, Public Health Service, Department of Health, Education, and Welfare, Atlanta.

Read before the 69th annual meeting of the American Society for Microbiology, Miami Beach, Fla, May 7, 1969.

Reprint requests to Special Pathogens Section, Bacterial Diseases Branch, Epidemiology Program, National Communicable Disease Center, Atlanta 30333 (Dr. Young).

Recovery of Bacillus Anthracis in Relation to Formaldehyde Vaporization

Building	Survey 1 Nov 8, 1967 Before Formaldehyde Vaporization	Survey 2 Dec 6, 1967 After Formaldehyde Vaporization	Survey 3 Sept 24, 1968 6 Mo of New Production
Carding area	16/43* (37%)†	8/100 (8%)†	1/100 (1%)
Spinning and related areas	7/56 (12.5%)‡	1/100 (1%)‡	0/100 (0%)
Weaving and related areas	3/43 (6.9%)	Not resampled	Not resampled

* Numbers on left of virgule indicate the number of positive surface swabs; those on right, total number of surface swabs taken in the building.
† $P < 0.005$.
‡ $P < 0.01$.

Results of the culture surveys are summarized in the Table.

The percent of positive recoveries is expressed in terms of number of culture plates showing one or more colonies of *B anthracis* relative to the total number of swabs taken in a given building. The reduction in contamination was highly significant in both the spinning ($P < 0.01$) and carding buildings ($P < 0.0005$).

These data do not take into account any quantitative differences in the number of *B anthracis* organisms per positive swab before and after formaldehyde treatment. On the first survey, very heavy contamination of the carding machinery was noted, eg one swab yielded 89 *B anthracis* colonies. After exposure of the working areas to formaldehyde there was marked reduction (tenfold to 100-fold) in the recovery of all bacterial flora, and the positive plates from the second and third surveys had only an occasional *B anthracis* colony. The one positive recovery of *B anthracis* (a single colony) in the final survey came from a window sill. All samples taken from machinery and active work areas after formaldehyde exposure were negative.

Twenty-four plates containing approximately 100,000 spores of an avirulent anthrax strain were placed at various points in the spinning room before treatment. After being exposed to formaldehyde for two days, three of these each contained two colonies; the other 21 were sterile.

Comment

Gaseous sterilization has been the subject of a comprehensive review by Phillips.[6] As outlined in his monograph, the only conceivable alternatives to formaldehyde would have been the use of ethylene oxide or β-propiolactone. Both have the significant disadvantage of being toxic, vesicant compounds for which there are no commercially available devices for large-scale dispersion. Ethylene oxide demands an exposure time of up to a day, and the area must be tightly sealed because of the compound's explosive flammability. The plant in question could not be made airtight.

The agent β-propiolactone is effective, rapidly sporicidal, and has been used successfully to decontaminate large enclosures in matter of hours.[7] However, β-propiolactone has been shown to be carcinogenic for certain animals.[6] While proof of this effect in man is lacking, this evidence plus its known irritating effects on contact or inhalation resulted in reluctance to use it in this large-scale operation. Vaporized formaldehyde was selected for this study because besides being simple and safe to use, it is relatively inexpensive.

The bactericidal and sporicidal qualities of formaldehyde have long been appreciated.[6] Raw wool and goat hair coming into the British Isles from areas where anthrax is indigenous is treated with formaldehyde before it leaves the dock. Similarly, dock facilities at certain US harbors and trucks used to transport imported raw goat hair to mills are periodically fumigated with vaporized formaldehyde. To our knowledge, however, large-scale decontamination of a manufacturing complex, such as this mill, with vaporized formaldehyde has not been reported, or for that matter has gaseous decontamination of rooms of the size (averaging almost 300,000 cu ft) treated here. This study shows that such a procedure coupled with cleaning operations significantly reduces *B anthracis* contamination, as measured by surface sampling techniques.

Vaporization of formaldehyde was car-

d///-

——CUT HERE——

HeKT

1360—McGraw-Hill—Warren: Tropical and Geographical Medicine 2/e—WG: D9623
Pack 203 (MV/2) L300,30,8" ⊞—Bin 0—01-26-89 17-13-54—OC—1122—Tape 695 jt

Ⓢ mb

CHAPTER 92 Anthrax · Philip S. Brachman

Anthrax, a zoonotic disease, has an interesting history dating from biblical times. Although not currently a major public health problem, it has been associated with focal, devastating epidemics; it has played a significant role in developmental microbiology; and was the first disease associated with its etiological agent. Development of specific and nonspecific preventive measures has resulted in a decline in incidence so that today anthrax occurs sporadically except for an occasional report of an epidemic and in a few countries where it remains endemic. The human disease appears in three forms. In the United States, approximately 95 percent of the cases are cutaneous anthrax and the remainder are inhalation anthrax; gastrointestinal anthrax cases are reported from other countries, in some more commonly than inhalation cases. Synonyms for anthrax include charbon, malignant pustule, Siberian ulcer, malignant edema, splenic fever, milzbrand, wool-sorter's disease, and ragpicker's disease.

PARASITE

Bacillus anthracis is a gram-positive, spore-forming, nonmotile bacillus (1 to 1.3 μm by 3 to 10 μm) that grows at 37°C on ordinary laboratory media [*1*]. Growth may be noted after 8 to 12 h and becomes characteristic after 18 to 36 h of incubation, revealing round, convex, grayish-white colonies 2 to 5 mm in diameter, which may show comma-shaped outshootings. Colonial tenacity, which is typical of *B. anthracis*, may be demonstrated by drawing the inoculating loop through the colony; the disturbed part should stand perpendicular to the surface of the agar and resemble beaten egg whites. Gram-stain preparation of artificial media growth reveals gram-positive, square-ended rods in long, parallel chains. Spore stains demonstrate central or paracentral spores that do not protrude beyond the outline of the bacillus. Direct fluorescent-antibody staining of organisms grown on bicarbonate agar in a 5% CO_2 atmosphere [2] and bacteriophage testing may be used to confirm the identification [*3*]. Agar-grown cells suspended in saline inoculated subcutaneously or intraperitoneally into guinea pigs, mice, or rabbits will cause death of the animal in from 24 to 72 h. Autopsy reveals evidence of general toxicity and hemorrhages in multiple organs. Animals inoculated subcutaneously will demonstrate subcutaneous gelatinous hemorrhagic edema at the inoculation site in addition to general toxemia. If broth cultures are used to produce the inoculum, in order to avoid nonspecific deaths, the organisms must be centrifuged, washed, and resuspended in saline before being inoculated into animals.

------CUT HERE------

——CUT HERE——

Hept

1362—McGraw-Hill—Warren: Tropical and Geographical Medicine 2/e—WG: D9623
Pack 203 (MV/2) L300,30,8" [5]—Bin 0—01-26-89 17-14-10—OC—1122—Tape 696 jt

In the oropharyngeal form, the initial lesion may be in the oropharynx or the organisms may be transported through the oral mucosa to the tonsillar or cervical lymph nodes, where they germinate, multiply, and produce toxin. The resultant lymphadenitis and associated edema may be so massive as to compress the respiratory passages.

Meningitis

Meningitis may be secondary to any of the above forms of anthrax infection. It results from hematogenous spread of bacilli from a primary focus. Rarely, a primary focus cannot be identified.

Toxin

Virulence of *Bacillus anthracis* is determined by a toxin and by capsular material, each coded by a different plasmid. The toxin consists of three components: edema factor, lethal factor, and protective antigen [6]. In human disease, sterilization of tissues with antibiotics may reduce the severity of the illness but the clinical course will continue until the toxin in the body has been metabolized or otherwise inactivated.

CLINICAL DISEASE

Cutaneous anthrax

Cutaneous anthrax usually occurs on exposed parts of the body, such as the face, neck, or arms. After an incubation period of 1 to 10 days (commonly 2 to 5 days), a round, small, pruritic, painless papule approximately 1.0 cm in diameter, is seen at the site of inoculation. Within several days a small vesicle, or a ring of vesicles, develops, surrounded by a small ring of erythema and slight, nonpitting edema. If multiple vesicles are present, they coalesce to form a single large vesicle. There may be lymphangitis and regional lymphadenopathy. Shortly thereafter, hemorrhage occurs at the base of the vesicle. The vesicle ruptures, discharging clear to slightly yellow serous fluid containing *B. anthracis* organisms. Beneath the vesicle is a well-demarcated, depressed ulcer crater, the base of which is covered with a developing black eschar. Over the next week as the eschar dries, it slowly separates from the surrounding tissue. The ulcer slowly granulates, leaving a small scar.

——CUT HERE——

———CUT HERE————————————————————————

HRKT

1364—McGraw-Hill—Warren: Tropical and Geographical Medicine 2/e—WG: D9623
Pack 203 (MV/2) L300,30,8" [I]—Bin 0—01-26-89 17-14-24—OC—1122—Tape 697 jt

Gastrointestinal anthrax

Gastrointestinal anthrax has an incubation period of 2 to 5 days. The initial symptoms of the abdominal form are nausea, vomiting, anorexia, and fever. As the disease progresses, significant abdominal pain develops and hematemesis and bloody diarrhea may occur. In some cases symptoms are severe and the patient appears to have an acute surgical abdomen. Ascites may be demonstrated on physical examination. Progression of the disease leads to toxemia, cyanosis, shock, and death, which may occur 2 to 5 days after the onset of the clinical disease. There has been a report of deaths occurring in less than 2 days after onset of the first symptoms (personal communication).

In oropharyngeal anthrax, the patient develops fever, anorexia, submandibular edema, and cervical lymphadenopathy [8]. In some reports acute inflammatory lesions resembling cutaneous lesions are described in the oral cavity involving the posterior pharyngeal wall, the hard palate, or tonsils. The edema of the cervical area may become so extensive that there may be encroachment of the oral passageways causing difficulty in breathing.

Therapy of gastrointestinal anthrax is the same as for inhalation anthrax. Additionally, tetracycline 1 g/day intravenously has also been reported to be effective. The fatality rate for gastrointestinal anthrax ~~exceeds 50 percent.~~ varies from 25 to 75 percent.

Meningitis

Anthrax meningitis symptomatically resembles other forms of acute bacterial meningitis. Therapy should be the same as for inhalation anthrax.

Immunity

Serological studies suggest that immunity develops after clinical disease and persists for a number of years. Reinfections have not been confirmed. There is some evidence to support the development of subclinical infections [9].

LABORATORY DIAGNOSIS

Laboratory diagnosis of cutaneous anthrax is made by culturing the vesicular fluid on ordinary laboratory media. In inhalation anthrax, sputum may be cultured; however, unless there is secondary anthrax pneumonia, cultures are negative. In gastrointestinal anthrax, vomitus or fecal material should be cultured; in anthrax meningitis, cerebrospinal fluid should be examined. In all forms of the disease, blood cultures may be positive. Fluorescent antibody staining and/or bacteriophage testing may be used to confirm the identification of *B. anthracis*. Serology can be used to demonstrate exposure to *B. anthracis*. The indirect hemagglutination test has been used but a more sensitive test is the ELISA test [10,11]. A recent development is an electrophoretic-immunotransblots method [11].

———CUT HERE————————————————————————

EPIDEMIOLOGY

Anthrax cases are classified as either industrial or agriculturally acquired [14]. In the United States, industrial anthrax accounts for approximately 80 percent of the cases. In other countries, agricultural anthrax is usually more common. An occasional case has been reported in which the source of infection is not discernible.

In the United States, the involved industries process imported goat hair, wool, skins and hides, bones, and bonemeal. Occasionally, the source of infections may be a commercial product such as animal yarn or novelties made from skins, hides, or animal hair that have not been properly disinfected. Cases sometimes occur among laboratory personnel.

Agricultural cases result from contact with carcasses of animals that have died of anthrax. Inadvertent inoculation of animal vaccine has been reported (but not documented) to cause cutaneous anthrax.

The route of transmission of cutaneous anthrax is primarily by direct contact, though occasionally indirect contact may be involved. Inhalation anthrax results from airborne transmission of organisms released into the air from equipment used to process the animal products, primarily goat hair or wool. Gastrointestinal anthrax results from eating inadequately cooked contaminated food, most often, meat.

The majority of cases are sporadic though occasional epidemics occur. In the United States the last epidemic occurred in 1957 and involved nine employees in a goat-hair processing mill; four were inhalation and five cutaneous cases [15]. All were traced to contact with a single batch of imported goat hair that appeared to be more heavily contaminated with *B. anthracis* than normal. During recent years, occasional epidemics have been reported in other countries, usually related to outbreaks of animal anthrax. An extensive epidemic occurred in Zimbabwe, which began in 1979 and by 1985 had abated; however, cases continue to occur, which may reflect the endemic occurrence. It is estimated that more than 10,000 cases, primarily cutaneous anthrax, have appeared Zimbabwe [16].

ED 1, in

Agriculturally related human cases parallel the existence of anthrax in the animal population. *Bacillus anthracis* spores are known for their resistance to chemical, physical, and environmental factors. They are reported to persist in nature for years, though this has not been proven under natural conditions. Anthrax districts may represent areas in which contamination persists for many years or areas that are reinfected at regular intervals by animals or other sources. These anthrax districts frequently contain alluvial soil with a pH of greater than 6.0.

Human-to-human or insect transmission has not been proven.

PREVENTION

Primary prevention of anthrax in humans involves controlling the disease in animals and preventing contamination of their products. This can be accomplished by practicing good animal husbandry, including immunization of animals at regular intervals using the Sterne strain vaccine and properly disposing of contaminated carcasses by means of deep burial or complete incineration. Animal products that are shown to be contaminated should be decontaminated with formaldehyde, ethylene oxide, pressurized steam, or gamma irradiation; they may also be discarded by burial or by incineration in a manner that does not result in contamination of the environment.

JOB 5369- 060-10 PAGE-009 TT-CHAPS 111-114
REVISED 25OCT88 AT 22:14 BY SD DEPTH: 0.00 PICAS

galley TT-[illegible] 10-20-88

[illegible]

Chapter 113
Anthrax

PHILIP S. BRACHMAN

Anthrax, a zoonotic disease, occurs in three forms in humans: cutaneous, accounting for 95% of cases seen in the United States; inhalation, accounting for 5%; and gastrointestinal, which has never been reported in the United States. The breakdown of cases throughout the world is probably similar; the few gastrointestinal cases reported have occurred in Asia and Africa. Meningitis and septicemia may be complications of any form.

ETIOLOGY

Bacillus anthracis is a gram-positive, nonmotile, capsulated bacillus ($1-1.3\ \mu m \times 3-10\ \mu m$) that produces central or paracentral oval spores which do not cause significant swelling of the rods. In smears from growth on ordinary artificial mediums, the bacilli lie in long, parallel chains. In clinical specimens, they occur singly or in short chains containing two or three square-ended or slightly rounded bacilli that are encapsulated. A specific fluorescent antibody conjugate stains the bacteria brilliantly.

The spores are formed under aerobic conditions, and they are relatively resistant to destruction by disinfectants and heat. They reportedly persist for years in the soil and in some animal products.

On ordinary culture mediums such as nutrient agar, after 18 hours at 37°C, colonies are round, approximately 5 mm in diameter, gray to white, slightly rough-textured, and with a ground-glass appearance. Comma-shaped outgrowths may project from the edge of the colony (medusa head or comet tail). Additionally, on 5% sheep blood agar, colonies are nonhemolytic.

The colonies of *B. anthracis* are tenacious; if an inoculating needle is drawn through a colony, the disturbed regions stand up like beaten egg whites. Capsule production may be helpful to presumptive identification in laboratories that do not have special reagents; if cultures are grown under increased carbon dioxide concentration on bicarbonate-containing mediums smooth, mucoid colonies result with *B. anthracis*. [illegible] by vegetative *B. anthracis*. Lysis of isolates by a specific anthrax gamma bacteriophage may be used to identify *B. anthracis* tentatively. Laboratory mice and guinea pigs die 2 to 5 days after inoculation with an agar-grown suspension or washed broth culture of *B. anthracis*.

EPIDEMIOLOGY

The average number of cases of anthrax reported annually in the United States has declined from 127 (1916–1925) to 0.7 (1977–1986) (Fig. 113–1). Of the 231 cases reported between 1955 and 1986, 20 were fatal.

F113-1

Page 9

49

AR100014

sions—most frequently in the terminal ileum or cecum—that may lead to hemorrhage. Extension to regional nodes may occur.

Oropharyngeal anthrax follows entry of spores of *B. anthracis* through the oral mucosa. Following deposition in cervical lymph nodes, the spores germinate, multiply, and produce toxin, causing inflammation of the infected area, local edema, and toxemia. The edema may be so severe that obstruction of the trachea results.

Bacillus anthracis produces a plasmid-mediated toxin consisting of three components: protective antigen, lethal factor, and edema factor. The virulence of *B. anthracis* is determined by two factors that are mediated by different plasmids: capsular material and toxin.

MANIFESTATIONS

Cutaneous Anthrax

After an incubation period of 1 to 7 days (usually 2–5 days), a small papule develops; the papule progresses to a vesicle over the next few days. The initial lesion may consist of a small ring of vesicles that coalesce to form a single large vesicle. Erythema and nonpitting edema may surround the vesicle. The initial symptom is usually pruritis without pain. The vesicular fluid is clear or slightly serous-colored, and initially contains large number of organisms. When the vesicle is ruptured, a sharp-walled, depressed ulcer crater with a black eschar developing in the center is revealed (Fig. 113–2A).

F113-2

There may be mild systemic symptoms, a degree or two of fever, malaise, and occasionally regional lymphangitis and lymphadenopathy. Further progression to general toxemia and septicemia is rare.

The typical eschar, when fully developed 7 to 10 days after onset, is round and 1 cm to 3 cm in diameter (Fig. 113–2B). With no secondary infection, the edges begin to separate from the crater. Eventually the eschar loosens and falls off. Healing continues by granulation, resulting in scar tissue.

F113-3

Lesions occur primarily on exposed parts of the body, such as the face, neck, and arms (Fig. 113–3). Rarely, multiple, simultaneously evolving cutaneous lesions have been reported. These probably are the result of simultaneous multiple inoculations.

Lesions in the periorbital area are frequently associated with extensive edema that may involve the entire face, extend down to the neck and upper chest, and impinge on the trachea. Similarly, lesions of the neck and upper chest may also give rise to extensive edema of the surrounding tissues.

"Malignant edema" is the term used to describe cutaneous anthrax associated with significant local reactions such as multiple bullae, extensive edema, induration, and with systemic illness resulting from general toxemia.

Inhalation Anthrax

Inhalation anthrax has a biphasic clinical pattern: the initial stage begins after an incubation period of 1 to 5 days as a nonspecific illness, with malaise, fatigue, myalgia, mild fever, nonproductive cough, and, infrequently, a sensation of precordial oppression. Rhonchi may be heard. The illness is frequently diagnosed as a respiratory infection. Within 2 to 4 days, symptoms may improve, but soon the second stage is heralded by the sudden development of severe respiratory distress, with dyspnea, cyanosis, stridor, and profuse diaphoresis. Subcutaneous edema of the chest and neck may develop. The pulse, respiratory rate, and temperature are elevated. There are moist, tests. The ELISA test should be run on two specimens of serum collected approximately 4 weeks apart. If a significant titer, or a rise in titer, is found, the electrophoretic immunotransblot test should be performed for confirmation. This test identifies antibody to the protective protein antigen and/or lethal factor protein. If these proteins are identified, the specimen is considered positive.

In anthrax meningitis, *B. anthracis* has always been recovered from the cerebrospinal fluid.

PROGNOSIS

Cutaneous anthrax, untreated, results in death in 10% to 20% of cases; with effective antimicrobial therapy, fewer than 1% of patients will die. Regardless of the kind or intensity of systemic antimicrobial therapy, cutaneous lesions progress through the classic changes. Adequate antimicrobial therapy, however, reduces local reactions, such as edema and erythema. A scar, proportional in size to the cutaneous lesion, will develop. Protective immunity appears to result, although there are reports of patients who have had two cutaneous infections, years apart. In none of these patients was there laboratory confirmation of both infections.

Inhalation anthrax is virtually always fatal, even with antibacterial therapy.

Gastrointestinal anthrax is associated with a 25% to ~~50%~~ fatality rate.

1–2 100%

The case–fatality ratio in cases of anthrax meningitis is also high, although nonfatal cases are occasionally reported.

Because antibody increases have been found in employees of goat hair mills who have no history of anthrax, subclinical infections must occur.

Page 13

THERAPY

Cultures must be taken within 24 hours of starting treatment for anthrax because specific therapy may inhibit the recovery of *B. anthracis*. The drug of choice in cutaneous anthrax is penicillin. In mild disease, peroral treatment with potassium penicillin V is suitable (30 mg/kg body wt/day, PO, in four equal portions, 6-hourly, for 5–7 days). With extensive lesions or in systemic illness, procaine penicillin G (20–30 mg [31,200–46,800 units]/kg body wt/day, IM, in two equal portions, 12-hourly, for 5–7 days) should be used. Many other agents are also effective, including tetracycline (15–20 mg/kg body wt/day, PO, in four equal portions, 6-hourly, for 5–7 days).

? Aw: Caution dosage please verify + initial

JJC PJB

69

Excision of cutaneous lesions is not recommended because it may lead to an intensification of the symptoms and possibly to the spread of infection. The local application of ointments containing antimicrobials has no effect. The cutaneous lesions should be kept clean and covered; soiled dressings should be bagged (in polyethylene) until incinerated. If hospitalized, the patient should be handled with drainage/secretion precautions. Glucosteroids (systemically) are said to reduce significantly the morbidity and mortality of severe cutaneous anthrax (malignant edema).

The therapy of inhalation anthrax is based on empirical knowledge and extrapolation from animal experiments. Massive doses of penicillin G by intravenous injection (50 mg [80,000 units]/kg body wt as a loading dose given in the first hour, with a maintenance dosage of 200 mg [320,000 units]/kg body wt/day) should be used. Streptomycin (7–15 mg/kg body wt as a loading dose and 15–30 mg/kg body wt/day as the maintenance dose, IV—to assure adequate concentrations in the blood) may also be used. Specific antitoxin may be of value; however, there is no do-

JJC PJB

Cap 18pt B

Journals

Albrink WS, Brooks SM, Biron RE, Kopel M: Human inhalation anthrax: A report of three fatal cases. Am J Pathol 36:457–471, 1960

Amidi S, Dutz W, Kohout E, Ronaghy HA: Anthrax in Iran. Z Tropenmed Parasitol 24:250–255, 1973

Brachman PS: Inhalation anthrax. Ann NY Acad Sci 353:83–93, 1980

Brachman PS, Fekety FR: Industrial anthrax. Ann NY Acad Sci 70:574–584, 1958

Brachman PS, Gold H, Plotkin SA, Fekety FR, Werrin M, Ingraham NR: Field evaluation of a human anthrax vaccine. Am J Public Health 52:632–645, 1962

Brachman PS, Plotkin SA, Bumford FH, Atchison MM: An epidemic of inhalation anthrax: The first in the twentieth century. II. Epidemiology. Am J Hyg 72:6–23, 1960

Dutz W, Kohout E: Anthrax. Pathol Annu 6:209–248, 197[illegible]

Glassman HN: World incidence of anthrax in man. Public Health 73: 22–24, 1958

Haight TH: Anthrax meningitis: Review of literature and report of two cases with autopsies. Am J Med Sci 224:57–69, 1952

Lincoln RE, Klein F, Walker JS, Haines BW, Jones WI, Mahlandt BG, Friedman RH: Successful treatment of rhesus monkeys for septicemic anthrax. Antimicrob Agents Chemother 4:759–763, 1964, 1965

Norman PS, Ray JG Jr, Brachman PS, Plotkin SA, Pagano JS: Serologic testing for anthrax antibodies in workers in a goat hair processing mill. Am J Hyg 72:32–37, 1960

Plotkin SA, Brachman PS, Utell M, Bumford FH, Atchison MM: An epidemic of inhalation anthrax: The first in the twentieth century. Am J Med 29:992–1001, 1960

Turnbull PCB: Thoroughly modern anthrax. Abst Hyg Trop Dis 61:R1–R13, 1986

Rep

Page 15

59